DISPERSION OF "REFRACTORINESS" IN PATIENTS WITH VENTRICULAR FIBRILLATION IN THE CHRONIC PHASE OF MYOCARDIAL INFARCTION.

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We have measured ventricular fibrillation(VF) intervals in pts with ventricular tachycardia/fibrillation(VT/VF) and in pts with VT, who underwent arrhythmic surgery. A control group without arrhythmia underwent aneurysmectomy. A balloon of 64 electrodes was inserted in the left ventricle and used for simultaneous endocardial recordings of VF intervals of 4 seconds duration at multiple sites and at normothermia. VF was induced by premature stimulation. At each recording site the interval between successive local depolarizations was measured. The averaged value, the so-called VF interval, was taken as index of local "refractoriness". Dispersion of "refractoriness" was defined as the variance (squared standard deviation) of the VF intervals at all sites.

	Mean VF Interval		Variance		
Control group	(n=4) 166.2±12.9 ms	ns	80.55	p<0.05	(df=115)
VT group	(n=3) 153.9± 4.7 ms	ns	53.59	p<0.0005	(df= 68)
VT/VF group	(n=4) 149.0± 3.9 ms	ns	209.03		(df= 69)

df: degrees of freedom

In conclusion: a) The variance in the VT group was significantly smaller than in the control group; b) The variance in the VF group was significantly larger than in the control group and VT group. Thus, dispersion in refractoriness is most pronounced in the clinical VF group.

ACUTE LEFT VENTRICULAR ISCHEMIA IMPAIRS RIGHT VENTRICULAR EARLY FILLING VIA THE SEPTUM

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We used pulsed Doppler to study RV peak early filling (RVE) and LV peak early filling (LVE) before and after 2 min of LAD occlusion in 11 dogs instrumented to measure LAP, LVP, RAP and RVP. We analyzed LVEDP, LVP minimum (LVPm), time constant of isovolumic LV relaxation (T), pressures at the mitral valve opening (MPCO) and tricuspid valve opening (TPCO), RVEDP and RVP minimum (RVPm). Results:

	Baseline	Ischemia
RVE (cm/s)	25.2± 9.9	17.5±10.0*
RVPm (mmHg)	3.7± 3.1	4.1± 4.5
RVEDP (mmHg)	7.4± 3.6	6.7± 3.1
TPCO (mmHg)	4.8± 3.1	4.9± 3.5
LVE (cm/s)	45.4± 12.0	32.9± 7.0*
LVPm (mmHg)	2.9± 2.7	5.8± 2.8*
LVEDP (mmHg)	7.4± 3.3	10.5± 3.9*
MPCO (mmHg)	8.3± 3.6	9.2± 3.5
T (ms)	36.6± 6.3	42.4±10.0*

(* p<0.05 paired t-test between ishemia and baseline)

In conclusion: in addition to the impairment of LV early filling, RV early filling during brief LV ischemia is significantly impaired due to LV-RV diastolic direct interaction via the septum.

Tuesday, March 5, 1991
Poster Displayed: 2:00PM–5:00PM
Author Present: 3:00PM–4:00PM
Hall F, West Concourse
Echocardiography

IMPORTANCE OF SAMPLING BOTH PULMONARY VEINS IN THE TRANSESOPHAGEAL ASSESSMENT OF SEVERITY OF MITRAL REGURGITATION

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Reversal of systolic flow (RSF) in the pulmonary veins (PV) by transesophageal echo (TEE) is useful in predicting severity of mitral regurgitation (MR). The issue of which pulmonary vein to sample in assessing RSF has not been established. We performed pulsed-Doppler of both the right and left upper PV in 56 patients (mean age 59 ± 14 years, 29 M) with MR grades 2-4+ by TEE color flow mapping. Measurements were PV peak systolic (S), diastolic (D) velocities (VEL), the presence of RSF, and jet direction (eccentric vs central).

MR Grade	n	Right PV cm/s S	D	n RSF	Left PV cm/s S	D	n RSF	Eccentric n	Jets	Central n
2+	10	55	47	0	53	49	0	4		6
3+	12	33	56	0	34	55	0	8		4
4+	34	1*	61	33*	12	61	22	25		9

*P ≤ 0.01, right vs left PV flow

Thirteen of 56 pts (23%) had discordant flow between the right and left PV (P=0.02). Of the 34 pts with 4+ MR, 11 (32%) showed discordance; all 11 had RSF in the right and not in the left PV; 22 (65%) had RSF in both PV while 0 pts had RSF in the left but not in the right PV. Of the 11 pts with discordance, 10 (91%) showed eccentric jets, antero-medial in 8 pts and postero-lateral in 2 pts, while 1 showed a central jet. Conclusions: 1) Discordant PV flow occurs between the right and left PV. 2) Left PV flow underestimates 4+ MR severity in 32% of pts, especially with eccentric jets. 3) Pulsed-Doppler of both PV is necessary to assess reversal of systolic flow as a marker of 4+ MR.

INTRACARDIAC SHUNTING ACROSS A PATENT FORAMEN OVALE MAY EXACERBATE HYPOXEMIA IN HIGH ALTITUDE PULMONARY EDEMA.

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Rapid ascent to altitude in susceptible individuals may result in severe hypoxemia, pulmonary hypertension, and even pulmonary edema. The underlying mechanism for this problem is unknown. We studied 12 climbers on Mt. McKinley, Alaska (4,200m) with bubble contrast echocardiography (Interspec XL) to determine whether the presence of a patent foramen ovale (PFO) might contribute to the pathophysiology of high altitude pulmonary edema (HAPE). Two of 2 climbers with HAPE, 1 of 2 with acute mountain sickness (AMS) but who did not have HAPE, and 2 of 8 well acclimatized climbers had documented right-to-left shunting at the atrial level (p=0.15 by Fisher's exact test for comparison between right-to-left shunting in climbers with AMS or HAPE, n=4; vs controls, n=8). Although the statistical power of these data is limited by the small number of observations, this study suggests that individuals with a PFO may be susceptible to right-to-left shunting during high altitude induced pulmonary hypertension. This phenomenon could result in a vicious cycle of worsening hypoxemia, and in lung regions with significant V/Q mismatch and low V/Q, increased alveolar hypoxia resulting in further hypoxic vasoconstriction and worsening pulmonary hypertension, thus exacerbating the disease process.